Journal of Biomedicine and Biosensors, 6(2):7–17, 2026. DOI: 10.58613/jbb622

Journal of Biomedicine and Biosensors

ISSN (online): 2583-3170

Journal homepage: https://gpub.org/journal-details.php?journal-id=45

Research Article

Inflammatory Biomarkers and Adiponectin in Post-COVID-19 Lung Fibrosis: Their Relationship with Disease Severity and Mortality

Maryam Kadhim Al-Shemery [1*], Ashraf Raoof Mohammed Ali[2] and Zahraa S.M Al Kaabi[1]

[1] *College of Science, Department of Pathological Analysis, University of Kufa. Iraq.*
[2] *College of Science, Department of Biology, University of Kufa. Iraq.*
[*] *Corresponding author: maryamk.alshemery@uokufa.edu.iq*

Article Info

Keywords: *Post-COVID, ESR, CRP, Adiponectin.*

Received: *10.05.2026;*
Accepted: *02.06.2026;*
Published: *10.06.2026*

Abstract

Lung fibrosis was most commonly lung problem induced in a post-COVID patient. The present research was demonstrated the linked between concentration of circulating CRP, ESR and adiponectin that associated with severity and mortality of post COVID-19 patient with lung fibrosis. The current study was conducted by taking 25 patients with lung fibrosis attending Al-Kafel hospital in the province of Karbala. It was carried out from the age of the patient's group was range from the 21-70 years. The information of patients was obtained through questionnaire consisted name, gender, age, weight, height. A group of 15 was considered healthy subjects. The result of current study showed there is significant increase in BMI in patients with PC (27.20 ± 0.9) when comparison to control. The result also, represented the percentage distribution of BMI of the patients with 24.57% normal weight, overweight 27.07% of the participants and about 29.9% suffered from obesity. The result in current study exhibits significant increase in inflammation characteristic (ESR and CRP concentration) in Post COVID 19 patients with lung fibrosis. Also, there is statistically significant increases in serum levels of Adiponectin (APN), in Post COVID 19 patients with Lung fibrosis in comparison with control. Conclusions: The present study offers an updated description of the biomolecule implications of APN in post COVID-19 with lung fibrosis and the high level of CRP to gather with elevated in ESR increased the risk for death in post COVID 19 patients with lung fibrosis. Elevated Adiponectin concentration was also correlation with an elevated rate of inflammation together with increase BMI.

1. Introduction

Coronavirus disease 2019 (COVID-19) is still increasing worldwide, and as a result, the number of patients with pulmonary fibrosis secondary to COVID-19 will expand over time [1]. Risk factors and comorbidities like advanced age with limited lung function, pre-existing diabetes, hypertension, cardiovascular diseases, and obesity have increased the risk for severe COVID-19 infection [2]. The classification is based on COVID-19 infection severity [3]. Stage 1 of disease characterized by mild symptoms, coughing, nasal congestion, high temperature, painful throat, muscle pains, overall discomfort, and migraine. While in second stage showed inflammation in pulmonary and coagulopathic events can occur, manifesting as dyspnea and hypoxemia [4, 5]. Some studies suggested that about 49% of the severe cases progress in acute respiratory distress syndrome (ARDS) and venous thromboembolism [5, 6]. One of the complications of infection is Pulmonary

How To Cite: M.K. Al-Shemery, A.R.M. Ali and Z.S.M. Al Kaabi. Inflammatory Biomarkers and Adiponectin in Post-COVID-19 Lung Fibrosis: Their Relationship with Disease Severity and Mortality. *Journal of Biomedicine and Biosensors*, 6(2):7–17, 2026. https://doi.org/10.58613/jbb622

fibrosis developed in third stage [5]. However, during this infection the emerging evidence points to a important role of persistent low-level inflammation linked to improperly functioning fat tissue enhances the inflammatory reaction [7]. Furthermore, the glucose and lipid metabolism regulated by adiponectin which exhibits anti-inflammatory features. So that, when the concentration of adiponectin decline reported in diabetes, obesity, nonalcoholic fatty liver disease and coronary heart disease [8–11].

Adiponectin (APN) is a type of adipokine that plays a role in managing glucose metabolism, enhancing insulin sensitivity, and facilitating the oxidation of fatty acids. It has a significant function in viral infections through the regulation of the immune response by balancing anti-inflammatory and pro-inflammatory pathways.

The low level of adiponectin causes intensifies the viral infections severity of due to adiponectin regulates immune cell activity by of inflammatory axis suppression and adenosine monophosphate protein kinase stimulation [12]. APN is polypeptide hormone that synthesis in adipose tissue, brain, placenta and skeletal muscles. It was first identified in 1995 in the different adipocytes by previous study [13], it was observed in mice adipocytes [14], however, in 2007 it was revealed that APN plays a role in adipocyte differentiation [15]. The gene of APN is presented on the chromosome region 3q27, which increases the susceptibility to type 2 diabetes mellitus (T2DM). The decline in concentration adiponectin levels has been linked to an elevated of type 2 diabetes mellitus risks. APN synthesis in adipose tissue is exhibits correlated to body mass index (BMI). Consequently, obesity or metabolic syndrome contribute to low level of APN concentration [16]. The different condition such as starvation as in anorexia nervosa caused APN plasma level is increase that release from adipose tissue with bone marrow [17]. Elevated plasma levels of adiponectin suppress the adipocyte differentiation and metabolic derangement, thereby elevated the insulin resistance (IR), type 2 diabetes mellitus (T2DM), and metabolic syndrome and metabolic syndrome [18]. Notably, APN induced the sensitivity of insulin by reduction of hepatic glucose synthesis and oxidation of peripheral fatty acid [16]. Previous study demonstrated that the elevation in molecular APN represents the most biologically active form involved in the regulation of glucose homeostasis [19]. Similarly, high molecular weight (HMW) APN is exhibiting a positive association with coronary artery disease [20]. APN concentration are elevated by aging, estrogen deficiency, and smoking [21]. This protein acts on precise receptors which are AdipoR1 (especially in skeletal muscles) predominantly located in skeletal muscles, AdipoR2 primarily expressed in the hepatic cell and T-cadherin-CDH13 (especially found in blood vessels and neurons cell) [22].

This study aimed to analyzed the linked between levels of C-reactive protein (CRP), ESR and adiponectin and the severity and mortality of post COVID-19 patient with lung fibrosis.

2. Methodology

2.1. Study Subject

The study was conducted at Al-Kafel hospital in the province of Karbala, Iraq.

2.2. Participants

Thirty - eight Iraqi individuals undergoing health assessments between January and June 2022 were included in the research. Additionally, twenty healthy adults without lung fibrotic (PC) symptoms associated with COVID-19 served as controls.

2.3. Inclusion and Exclusion Criteria

Participants contributed in the study underwent health assessments and met the following criteria: they were between 21 and 70 years of age and undergoing lung examinations for medical reasons. Participants were excluded if they had a history of Idiopathic Pulmonary Fibrosis, Silicosis, Asbestosis, Rheumatoid Arthritis, Systemic Lupus Erythematosus, Tuberculosis, Asthma and hepatitis, if they were pregnant or lactating, or if they smokers.

2.4. Assessment criteria

Participants underwent comprehensive health assessments, including (ESR, CRP, Adiponectin).

Thirty-eight adult participants diagnosed with post-COVID-19 pulmonary fibrosis were recruited for this study. Diagnosis was confirmed based on established clinical and radiological criteria for post-COVID interstitial lung disease, including high-resolution computed tomography (HRCT) findings consistent with fibrotic changes, in accordance with current ATS/ERS guidelines. Individuals with pre-existing interstitial lung disease or other organic intestinal disorders were excluded. All participants were required to complete self-administered diagnostic questionnaires [23].

2.5. BMI Calculation

Body mass index (BMI) was analyzed by dividing the individual's self-reported body weight (kg) by their height squared (m). BMI was classified based on the WHO physical status classification17: (BMI $<$18.5 kg/m^2) considered underweight, BMI 18.5-25 kg/m^2 was normal weight, BMI 25-30 kg/m^2) was overweight (or obese (BMI $>$30 kg/m^2) [24].

2.6. Laboratory Analyses

Blood samples were collected via venipuncture using sterile syringes of 5mL. Each sample was immediately transferred into designated tubes. The blood was left to clot at room temperature for 10 minutes, followed by centrifugation at 6000 rpm for 15 minutes. The resulting serum was then stored frozen at -80 ℃ for subsequent laboratory analysis.

Erythrocyte sedimentation rate (ESR) was determined using the standard Westergren method [25, 26]. Venous blood samples (approximately 2 mL) were collected into tubes containing sodium citrate as an anticoagulant. The sample was gently mixed and aspirated into

a calibrated Westergren tube (200 mm). The tube was placed vertically in a rack at room temperature (18–25°C). After 60 minutes, the distance (in millimeters) that erythrocytes had fallen was measured and reported as mm/hour [27].

Serum C-reactive protein (CRP) levels were testing by an automated analyzer based on a latex-enhanced immunoturbidimetric assay. Venous bood specimens were gathered in uncoated tubes that did not contain any anticoagulant and spun at 3000 revolutions per minute for a duration of 10 minutes to separate the serum. The assay is based on the reaction between CRP in the sample and specific anti-CRP antibodies coated on latex particles, leading to agglutination proportional to CRP concentration. The intensity of turbidity was measured photometrically, and results were expressed in mg/L according to the manufacturer's instructions [28].

Serum adiponectin level was analyzed by an (ELISA) kit (Elabscience, USA) according to the manufacturer's instructions. The blood samples were collected and centrifuged to obtain serum, which was then stored under appropriate conditions until analysis. All reagents and samples were brought to room temperature prior to use. In summary, standards and serum samples were introduced to microplate wells pre-coated with specific antibodies against adiponectin. Following an incubation period, unbound substances were removed by washing. A biotinylated detection antibody specific for adiponectin was added, followed by streptavidin–horseradish peroxidase (HRP) solution. After a further incubation and washing step, substrate solution was added to develop color. The process was halted with a stopping solution, and the absorbance was recorded with a microplate reader at the recommended wavelength. Adiponectin concentrations were.

2.7. Statistical Analysis

The information was examined utilizing the Statistical Package for Social Sciences (SPSS) software version 26.0. Significance testing involved calculating descriptive statistics such as means and standard deviations for comparisons between patient and control subgroups. Data visualizations were created using Microsoft Office 2016 Excel. All statistical analyses were conducted with a significance level set at $P < 0.05$.

3. Results

3.1. Patient Characteristics

In this research, a total of 40 cases consisting of 25 post-COVID-19 patients who develop lung fibrotic (PC) and 15 controls were examined. As shown in Table 1, The patients had ages ranging between 21 to 70 years. As illustrated in same table, there is significant increase in BMI in patients with PC (27.20 ± 0.9) when comparison to control. The result also, represented the percentage distribution of BMI of the patients with 24.57% normal weight, overweight 27.07% of the participants and about 29.9% suffered from obesity. In this study, it has been shown that equal percentage distribution of gender Table 1.

Table 1: Clinical features of patients who develop lung fibrosis after COVID-19 compared to control group

Clinical characteristics	**Mean ± SE**	
	Patient N=38	**Control N=20**
Age (year)	54.0714 ± 4.18	63.1667 ± 9.19
BMI (kg/m2)	27.20 ± 0.9 *	21.94 ± 1.18
Normal weight	22.57 ± 0.8 (24.5 %)	
Overweight	27.07 ± 0.6 (27.5%)	
Obese	30.91 ± 0.8 (29.9 %)	
Gender	Male (50%)	
	Female (50%)	
Vaccinated	Yes (71.4%)	
	No (28.6%)	

* $P<0.05$ statistically significant with control group

The Figure 1 shown the specific CR test levels between the studied groups According to this figure there was a significant increase ($p<0.05$) of CR level in patients with post-COVID-19 Patients Who Develop Lung Fibrotic (PC) comparison with healthy group.

However, the Figure 2 illustrates the specific hematological test levels among the studied groups It reveals a significant elevation ($p<0.05$) of ESR level in patients with lung fibrosis when compared to healthy group.

3.2. Evaluation of biomarkers associated with PF

Adiponectin levels were assessed as a specific biomarker between the studied groups. Our data showed a significant elevated ($p <0.05$) in Adiponectin levels in patients diagnosed with pulmonary fibrosis compared to the healthy group Figure 3.

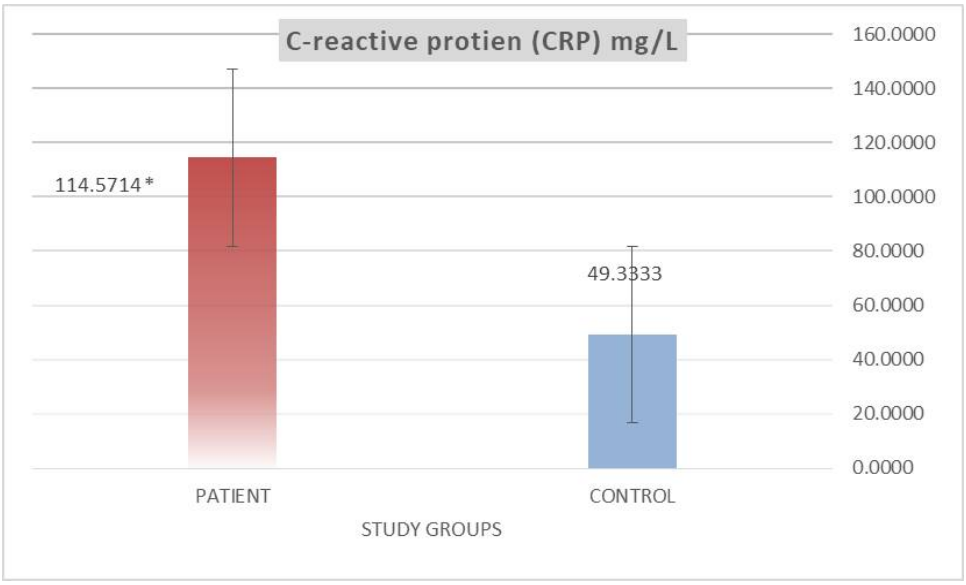

Figure 1: Comparison of the C reactive protein CRP between Groups of Post-COVID-19 Patients Who Develop Lung Fibrotic and control group * P <0.05 statistically significant with control group

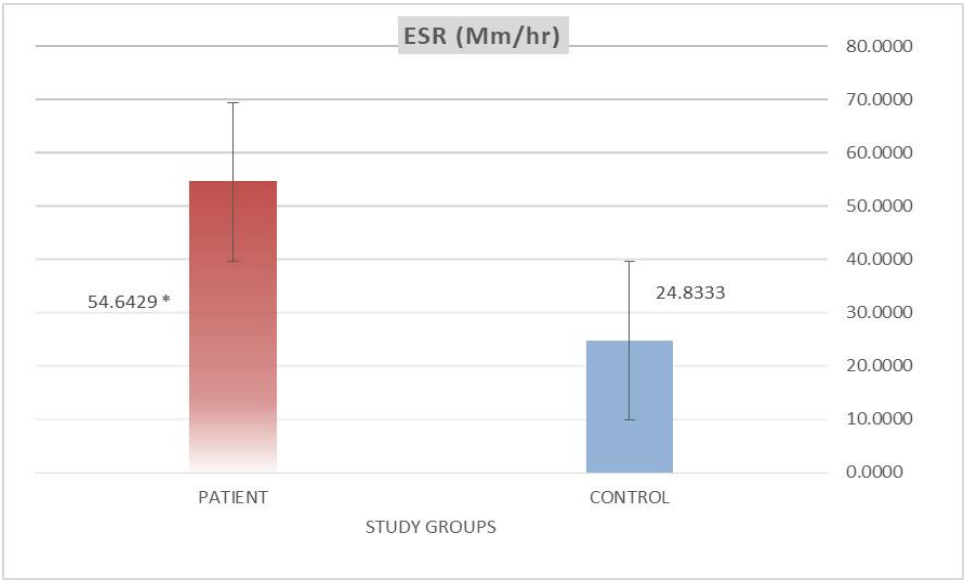

Figure 2: Adiponectin levels in the healthy and post-COVID-19 patient groups that develop lung fibrosis * P<0.05 statistically significant with control group

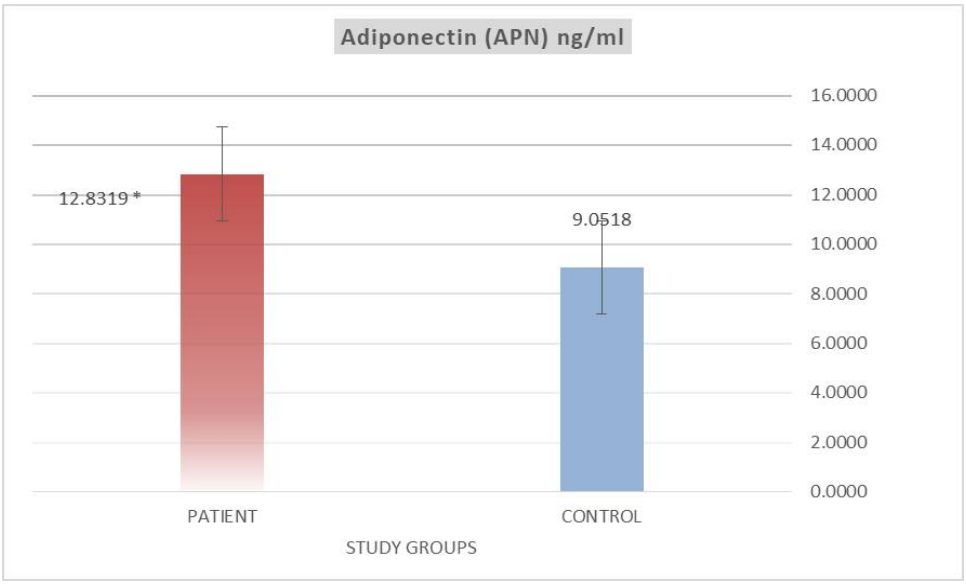

Figure 3: Comparison of the ESR between Groups of Post-COVID-19 Patients Who Develop Lung Fibrotic and healthy group

4. Discussion

Following infection with COVID-19, a subset of patients develops persistent pulmonary abnormalities, including interstitial changes that may progress to Pulmonary Fibrosis. This condition is mainly observed after severe acute illness and is associated with diffuse alveolar damage, dysregulated inflammatory responses, and abnormal tissue repair leading to fibroblast activation and excessive extracellular matrix deposition [29, 30]. Persistent inflammation and impaired resolution of lung injury have been proposed as key mechanisms driving fibrotic remodeling in susceptible individuals [31]. Risk factors include advanced age, severity of initial infection, prolonged mechanical ventilation, and elevated markers of inflammation such as CRP and IL-6 during the acute phase [32]. However, radiological and functional improvement may occur over time in a proportion of patients, suggesting partial reversibility in early fibrotic changes.

Numerous studies are conducted to obtain more information about post-COVID 19 pulmonary fibrosis. Elderly precipitate caused higher risk for lung fibrosis development after COVID-19 infection. Another risk factor such as diabetes, hypertension, lymphopenia and leukocytosis increased the disease severity [33].

The result of this research showed there is significant elevated in (CRP) level in patients with Lung Fibrotic following COVID-19 infection in comparison to healthy group. These findings were consistent with Colarusso [34] who found plasma CRP was statistically elevation in patients than in healthy subjects. SARS-CoV-2 infected patients develop a systemic inflammatory syndrome [35]. C reactive protein was inflammatory marker system connected with the severity of the infection [36]. Our report closely with those reported by the findings of Yilmaz [37] who observed that inflammation parameters during the early stages following hospital admission. Specifically, markers such as CRP, ESR, and PCT were substantially elevated, while Lys levels were notably significantly decreased. Furthermore, the previous study demonstrated that the inflammatory reactions appeared to markedly greater in COVID-19 patients with pulmonary fibrosis than without [38]. Since COVID-19 widely associated with severe inflammation, such as inflammatory storm. Consistent with previous researches [39–41] Nair found elevated CRP were linked to pulmonary fibrosis [33].

In our study, we found that the high level of C-reactive protein is an indicator for post-COVID-19 pulmonary fibrosis that agree with study documenting that CRP levels correlated with the level of inflammation, and its level was not affected by factors, such as sex, age, and physical condition [42]. Previous study shown, CRP is an easily available and universally acceptable inflammatory marker and documented to play a very crucial role in predicting timings of interventions and post-COVID lung fibrosis [43]. In COVID-19, pneumonia pathophysiology includes activation immune system, inflammation, thrombogenicity, and direct viral affection to the lungs and extrapulmonary tissues [44]. In COVID-19 pneumonia, CRP can be utilized as an indicator of inflammation that can used to analyze be utilized to analyze both infectious and noninfectious causes, as well as postoperative, surgical, venous thromboembolism, gout, and rheumatoid arthritis, as well as infectious and noninfectious causes [45, 46]. There is information on CRP in severe H1N1 viral pneumonia [47], and a several recent studies have shown a correlation between C-Reactive Protein and severity of COVID-19 disease [48–52] The recent study demonstrated that highly circulating CRP concentration causes the IPF risk factors of elevated in people of European descent [53]. Because, at sites of inflammation macrophages was induced by this inflammatory marker by released tumor necrosis factor and interleukin-1 [54], which regulates activation of fibroblast which production of scar tissue by angiogenesis and extracellular matrix deposition [55]. However, C-Reactive Protein causes significantly promotion fibrosis in organ with various organs. Previous study relived that C-Reactive Protein contributed to fibrosis of kidney through mechanism of TGF β/Smad3 pathway [56], but other study demonstrated that under high Ang II conditions, C-Reactive Protein could cause cardiac fibrosis by activating the TGF-β/Smad and NF-kB signaling axis in high Ang II conditions [57]. This axis also, has a main role in fibrosis of pulmonary which the reduction in promoting of TGF β/Smad3 can decline the progression of lung fibrosis [58, 59]. Consequently, the high concentration of this marker causes elevate the lung fibrosis risk of by influencing the axis connected with lung fibrosis.

The result of current research showed there is significant elevation in (ESR) level in patients with PC in comparison to healthy group. These findings were consistent with [60] who found that the risk of fibrosis was proportional relationship with ESR, CRP, and LDH, as well as with the length of hospital stay.

Recent study found that among all inflammatory markers, ESR level demonstrated as an important marker for fibrosis in lung prediction in patients with COVID-19. Other study showed that ferritin, LDH and CRP have no discernible correlation HRCT pulmonary fibrosis [61]. Strangely, Pu [62] relived that the laboratory result in this case showed the ESR level significantly higher after 2 wk infection with COVID-19. Even though the dry cough and fever subsided, improved the chest CT scan manifestations, and the test of throat swab nucleic acid assay came back negative, the ESR level remained increased for a considerable amount of time. This study showed the increased of ESR is abnormal in this case which do not induce due to the inflammation, cancer, TB, hyperthyroidism, rheumatoid, autoimmune diseases, anemia or drugs [62].

The erythrocyte sedimentation rate (ESR) is influenced by factors such as the shape and size, levels of red blood cells, as well as the properties of plasma [63]. The reasons for the ESR elevated in this instance remain uncertain. It is hypothesized that COVID-19 could potentially induce changes in the structure of erythrocytes or the composition of plasma. The prognosis of COVID-19 patients may be negatively impacted by a persistently elevated ESR, since high ESR could damage the joint and thus leads to joint diseases such as osteoarthritis [64, 65]. Furthermore, it may be a precursor of hepatic and renal dysfunction [66]. Studies are reporting that the increased sedimentation is more pronounced in severe diseases. A study assessing data from 148 confirmed COVID-19 patients, performed in Turkey by Kaya et al., found that median ESR was significantly higher in patients with severe/critical disease (66.5 compared to 35.5, $p < 0.001$). In this study, ESR was identified to be an independent factor in predicting severe disease and death, with the cutoff value of 52.5 mm/h having Sensitivity and specificity for severe disease were 65.5 percent and 76.3 percent, while the cutoff value of 56.5 mm/h had 66.7 percent and 72.5 percent for mortality [67]. The previous research comprised 358 out of 819 patients in total classified as serious situations, observed that in spite of all the diversity and constraints of the research showed increased in (ESR) was correlated with serious cases of COVID-19 in contrasted to non-severe cases. Although, the diagnostical and analytic was low, the findings indicated that monitoring ESR could prove useful in tracking progression of disease. [68]. The high level of ESR connected to elevation of serum proteins in diseases related to inflammation, infection, and cancer. So that, anemia, female gender, pregnancy, age, high BMI, and chronic illnesses all increase the test's low specificity [69].

However, the formulas contended Sex and age are frequently advised and utilized for determined normal test of ESR [70]. Erythrocyte sedimentation rate and C-reactive protein tests are frequently requested in tandem in clinical practice. Furthermore, due to shorter half-life of

CRP was more rapidly decline with the resolution of inflammation which is useful for quickly evaluating the effectiveness of treatment. These testes are importantly associated, while discrepancies between the two measures can occasionally occur under certain conditions. [71]. A meta-analysis demonstrated that utilized commonly the two markers to tested different acute inflammatory situations. Although other results were inconsistent, the diagnostic contributions remained comparable but utilizing both methods in conjunction was found to enhance diagnostic accuracy [72]. However, the result aimed to analyzed the usefulness of ESR for recognizing the severe from non-severe COVID-19 cases. [73]. ESR levels were elevated in COVID-19 cases have pneumonia and severe disease; so that, it was not indicative of any predictions [74].

The current research showed there is significantly elevation in (APN) level in patients with lung fibrosis in comparison to control group. An endocrine organ is the adipose tissue, released several adipokine and affected the secreted of inflammatory mediators and such as the substances comprised to a state of inflammatory system and causes altered pulmonary function either directly or systemically [75–77]. Increased lung inflammation from environmental exposure, increased susceptibility to lung infections, and exacerbation of airway obstruction in preexisting lung diseases may all be influenced by increased adipose tissue [78] Hindsberger [79] adiponectin Circulation level at the time of hospital admission, respiratory failure and mortality in SARS-CoV-2 infection were inversely correlated with admission and more researches are required to clarify the specific mechanisms through which adiponectin are connected to the course and outcome of COVID-19 [79].

When compared to healthy individuals, COVID-19 patients had significantly higher levels of insulin, leptin, and adiponectin ($P \leq 0.01$). All COVID-19 patients were obese and had severe respiratory inflammation, which may have led to elevated levels of insulin, leptin, and adiponectin [80]. Because adiponectin negatively affects the sodium balance in tissue ACE2 expression and pulmonary viral load, it is reduced in response to low sodium diet although angiotensin II infusion. The mechanism of obesity which is associated with the risk of severe COVID-19 infections may be explained by the imbalanced production of adiponectin [81, 82]. Adiponectin released from the adipose tissue in three forms related to their molecular weight (low, medium, high) that associated negatively with the amount of the adipose tissue [83]. In other study, the low circulated adiponectin concentration may not connect to the cardiovascular risk. Although, adiponectin levels low in response to the decrease in sodium diet and angiotensin II infusion [84]. So that, more researches demonstrated that serum levels of calcium, potassium, and sodium decreased in individuals with serious infection of COVID-19 [82–85].

Furthermore, several factors such as high BMI, Type 2 DM and metabolism syndromes caused the adipose tissue dysregulated, with the production of different cytokines that decrease the adiponectin level [86, 87]. Acute and chronic phases of different infections of virus have been shown to contain adiponectin. With its anti-inflammatory/pro-inflammatory pathway, it plays a part in the immunity regulation of virus infections. As a result, low levels of this hormone causes elevated the severity of disease and are associated with the stage of necro-inflammatory in cases of hepatitis B and C [88]. Consequently, adiponectin concentration could be elevated in mild to moderate COVID-19 individuals as a defense against SARS-CoV-2 by inflammatory pathway induced, and reduction in COVID-19 patient with severe symptoms due to disease-related dysfunction of fat tissue with reduced production of this hormone [89].

5. Conclusion

In conclusion, the current research highlights the importance of inflammatory and metabolic biomarkers in post-COVID-19 lung fibrosis. Elevated inflammatory markers, particularly CRP and ESR, were associated with increased disease severity and poorer outcomes, reflecting ongoing systemic inflammation and its role in fibrotic progression. In addition, adiponectin showed a significant linked to post-COVID-19 pulmonary fibrosis, suggesting a potential involvement in inflammatory regulation and tissue remodeling. Higher levels of these biomarkers may indicate enhanced inflammatory activity and increased risk of adverse clinical outcomes, although the exact mechanisms remain unclear and require further investigation.

Article Information

Acknowledgments: The authors thank the staff of Al-Kafel hospital in the province of Karbala, Iraq. Also, we would like to thank and appreciate Iraqi citizens' facilities for carrying out this study. Also, the authors would like to thank the Department of Pathological Analysis, College of Science, University of Kufa. Iraq. For their generous support for this study.

Author Contributions: M.K.A - Conceptualization, Methodology, Formal analysis, Writing – original draft, Writing – review & editing, Supervision; A.R.M.A - Formal analysis, Writing – review & editing; Z.S.M.A - Data curation, Writing – original draft, Writing – review & editing.

Funding / Financial Support: The authors received no external funding.

Conflict of Interest: The authors declare no competing interests.

Ethical Approval: The study was approved by the Ethical Committee of the University of Kufa, Iraq. All procedures involving human participants were performed in accordance with the ethical standards of the institutional research committee and the Helsinki Declaration.

Informed Consent: Informed consent was obtained from all participants involved in the study.

Data Availability Statement: The data supporting the findings of this study are available from the corresponding author.

Clinical Trial Registration: Not applicable.

Reporting Guidelines Statement: This observational study was conducted in accordance with the STROBE reporting guidelines.

Disclaimer (Artificial Intelligence): The author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.), and text-to-image generators have been used during writing or editing of manuscripts.

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